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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/633,789	08/04/2003	Curtis C. Harris	015280-225111US	6897

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EXAMINER	
GUPTA, ANISH	
ART UNIT	**PAPER NUMBER**
1654	

MAIL DATE	DELIVERY MODE
06/07/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/633,789	HARRIS ET AL.	
	Examiner	Art Unit	
	Anish Gupta	1654	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *21 March 2007*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1 and 16-24* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1 and 16-24* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. Applicants amendment, filed 3-21-07, is acknowledged. Claims 1 and 18 were amended. Claims 1, 16-24 are pending in this Application.

2. Applicant's election of Group I in the reply filed on 8-1-06 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)).

Applicant canceled claims 2-9 and 11-15 which were restricted in the previous office action into Groups II-V. Claims 16-24 were added. Claims 1, 16-24 are pending in this Application.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the applicant for patent.

The changes made to 35 U.S.C. 102(e) by the American Inventors Protection Act of 1999 (AIPA) and the Intellectual Property and High Technology Technical Amendments Act of 2002 do not apply when the reference is a U.S. patent resulting directly or indirectly from an international application filed before November 29, 2000. Therefore, the prior art date of the reference is determined under 35 U.S.C. 102(e) prior to the amendment by the AIPA (pre-AIPA 35 U.S.C. 102(e)).

3. Claims 1, 16-24 are rejected under 102(e) as being anticipated by White et al. (US 5604113).

The claims are drawn to a method for screening a compound for an ability to modulate apoptosis.

The reference of White et al. teach a method of identifying compounds and compositions that interact with putative onogenes by testing the ability of these compounds to suppress teh p53 mediated actions of the putative oncogene (see col. 13, lines 7-11). The reference states that the test compound is added to cell that express (i)a gene product that induces p53 mediated apoptosis; (ii) a gene product for a p53 gene, wherein either the gene or the gene product are externally controllable; and (iii) a putative oncogene that inhibits the effect of the gene product that induces p53 mediated apoptosis, and (B) examine said cells to determine whether apoptosis has occurred or proliferation has been controlled or induced. For example, 10 ul of a 1 mg/ml solution of 96 compounds can be added to such cells grown and maintained at the permissive temperature in a 96-well microtiter plate. (Other concentrations may be used, based on what is known about cytotoxicity of each compound or composition.) Apoptosis will typically occur in 24-48 hours and requires minimal intervention. If the test compounds cause cells to die or to cease proliferating, this may be due to p53-mediated events, or to general cytotoxicity. The compounds or compositions that have an effect would be further tested by serially diluting the compound to determine that minimally effective concentration (see col. 13, lines 11-30). This disclosure meets the limitation of the claims because the reference disclose all of active method steps, i.e. the addition of the test compound to a cell and determination if the test compound modulates apoptotsis. Although the reference does not teach helicase XPB or XPD or inhibition of binding of p53 to the helicase, such activity would inherently be present since the reference disclose death of cells are due to p53 mediated events.

Response to Arguments

Applicants state that White et al. "is devoid of any inherent disclose regarding helicases or inhibition of helicase binding to p53." Applicants state that prior to the instant specification it was unknown that p53 apoptosis was helicase dependent. Based on White et al. "one of skill in the art would have no technical bases to conclude the inhibition f helicase binding to p53 could modulate p53-mediated apoptosis. . . .White et al. does not make it clear to one of skill in the art that that [*sic.*] modulators of helicase-dependent p53-mediated apoptosis could be identified by detecting whether a test compound can inhibit p53 binding to helicase as required by the present claims."

Applicants arguments have been fully consider but have not been found persuasive.

First, Applicants have not argued that the reference does not meet all of the active method steps, i.e., the addition of the test compound to a cell and determination if the test compound modulates apoptotsis. Further, since the reference disclose an assay using cells in the assay, the helicase would necessarily be present. Thus, the active method steps disclosed in the prior art meet the claimed limitation. Applicants argue that the reference does not disclose the specific mechanism by which p53-mediated apoptosis, i.e. helicase dependent p53-mediated apoptosis. However, since White et al. disclose p53-mediated apoptosis, which Applicants do not argue, the reference disclose the basis of the inherency, since the mechanism of p53 apoptosis would be the same. It is the premise of the rejection that only one mechanism is involved in P-53 mediated apoptosis. Since the reference discloses determination if the test compound modulates p53-mediated apoptotsis, the reference meets the limitation of the claims and inherently meets the limitation of XPB and XPD helicase activity. Applicants can overcome this rejection by a showing that the art recognized other mechanisms that mediate p53 apoptosis.

Rejection is maintained.

4. Claims 1, 16-24 are rejected under 102(e) as being anticipated by Reed et al. (US 5484710).

The claims are drawn to a method for screening a compound for an ability to modulate apoptosis.

Reed et al. teach screening assay for identifying agents that inhibit p53 mediated regulation of a gent containing the p53-RE and thus can reduce of inhibit apoptosis in a cell (see col. 16, example V). The reference also disclose assay methods for identifying agents that can act as p53 analogs and can induce apoptosis in a cell (see example IV, col. 15). For methods to identify p53 analogs, the cells utilize include p-53 null cell lines or tumor cell lines that express mutant p53 gene and is obtained from cancer patients (see col. 15 and 16). For methods involving agents that inhibit apoptosis in cells, the reference states that the cell can be either 1) a cell that is obtained, for example, from the American Tissue Type Culture and is known to exhibit the characteristics of a cell obtained from a patient having a particular disease such as ataxia telangiectasia or 2) a neuronal cell line such as the cell lines described by Behl et al. (1993) that is exposed, for example, to amyloid beta protein (ABP) or to glutamate and, therefore, is a model for the type of cell death that occurs in Alzheimer's disease or in stroke, respectively. In this case, the assay provides the advantage that the cell lines that are used in the assay are adapted for tissue culture (see col. 18). This disclosure meets the limitation of the claims because the reference disclose all of active method steps, i.e. the addition of the test compound to a cell and determination if the test compound modulates apoptotsis. Although the reference does not teach helicase XPB or XPD or inhibition of binding of p53 to the helicase, such activity would inherently be present since the reference disclose death of cells are due to p53 mediated events.

Response to Arguments

Applicants state that Reed et al. does not provide a technical basis for one of skill in the art to conclude that inhibition of helicases binding to p53 could modulate p53-mediated apoptosis. "Without the teaching of the instant specification, one skill in the art would not be able to extrapolate form Reed et al.'s disclose that because binding of p53 to particular response elements can modulate p5 induced apoptosis, p53 must necessarily bind to helicase."

Applicants arguments have been fully consider but have not been found persuasive.

First, Applicants have not argued that the reference does not meet all of the active method steps, i.e., the addition of the test compound to a cell and determination if the test compound modulates apoptotsis. Further, since the reference disclose an assay using cell lines, the helicase would necessarily be present. Thus, the active method steps disclosed in the prior art meet the claimed limitation. Applicants argue that the reference does not disclose the specific mechanism by which p53-mediated apoptosis, i.e. helicase dependent p53-mediated apoptosis. However, since the reference disclose p53-mediated apoptosis, the mechanism by which apoptosis occurs would have to be the same. It is the premise of the rejection that only one mechanism is involved in P-52 mediated apoptosis. Since the reference disclose determination if the test compound modulates p53-mediated apoptotsis, the reference meets the limitation of the claims and inherently meets the limitation of XPB and XPD helicase activity. Applicants can overcome this rejection by a showing that the art recognized other mechanisms that mediate p53 apoptosis.

5. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

6. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Anish Gupta whose telephone number is (571)272-0965. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Cecilia Tsang, can normally be reached on (571) 272-0562. The fax phone number of this group is (571)-273-8300.

6/1/07
ANISH GUPTA
PRIMARY EXAMINER